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Stress-related changes in financial risk taking: Considering joint effects of cortisol and affect

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Abstract

Many decisions under risk and uncertainty are made under physical or emotional stress. A recent meta-analysis suggested that stress reliably influences risk taking but did not find a relation between single measures of stress such as cortisol and risk taking. One reason for the conflicting findings could be that the influence of stress on risk taking depends not only on physiological but also on psychological stress responses, in particular affective valence. We tested this hypothesis in an exploratory empirical study: Seventy participants worked on a financial risk-taking task. In half of the participants acute stress was induced with a cold pressor task. For all participants we measured cortisol and α-amylase levels, blood pressure, subjective arousal, and affective valence before and after the task. The stress induction increased participants' levels of cortisol, subjective arousal, and systolic blood pressure but did not directly influence negative affect or risky decision making. Examining the interplay between physiological and psychological stress responses, a moderation analysis revealed an interaction between stress induction and affect valence: Negative affect predicted an increase in risk-seeking decision making in the stress condition, but not in the control group. A similar moderation was found with cortisol reactivity, that is, negative affect predicted an increase in risk-seeking decision making in participants with high cortisol reactivity but not in participants with low cortisol reactivity. These results suggest that the effect of stress on risky decision making depends on the interplay of affective valence and cortisol reactivity.

KEYWORDS

acute stress induction, affect, cortisol, decision making, risky choice

1 | INTRODUCTION

In the past decade research on the influence of stress on risky decision making has surged. Overall, the results suggest a small but reliable effect, indicating that stress increases risk-taking behavior (Starcke & Brand, 2016). However, the pattern of results is highly heterogeneous. While some studies investigating the effect of stress on financial risk taking have reported increases (e.g., Buckert, Schwieren, Kudielka, & Fiebach, 2014; Starcke, Wolf, Markowitsch, & Brand, 2008), others have reported decreases in risky decision making (e.g., Cahlíková & Cingl, 2017; Pabst, Brand, & Wolf, 2013a) or

no effect (e.g., Sokol-Hessner, Raio, Gottesman, Lackovic, & Phelps, 2016; von Dawans, Fischbacher, Kirschbaum, Fehr, & Heinrichs, 2012). In the current research we investigate whether the contradictory findings can be reconciled by considering the heterogeneous nature of stress responses. Stress responses involve a range of physiological and psychological reactions that can occur together but can also show different paths of activations (Dickerson & Kemeny, 2004). Often research has focused on a single response, but whether stress impacts risky decision making may depend on the interplay of physiological and psychological responses elicited.

1.1 | Physiological and psychological responses to acute stress

Stress can be defined as an individual's response to a stressor, that is, an environmental condition or task that challenges or even exceeds the resources of the individual. Stressors can be of a physical nature, such as heat, cold, or pain, or psychological, such as the threat of exclusion or failure (Lazarus, 2006). Typical stressors employed in decision research include the cold pressor task (CPT; Lovallo, 1975), a physical stressor where participants immerse their hand and arm in ice water for up to 3 min, and the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993), in which participants have to endure a demanding evaluative social situation. More recently, a combination of a physical and a social stressor, the social evaluative cold pressure task (SECPT) was developed. In the SECPT participants are filmed while they immerse their hand in ice water (Schwabe, Haddad, & Schächinger, 2008). Stress responses include a variety of physiological and psychological reactions. On the *physiological level*, acute stress involves activation of the autonomous nervous system, causing a fast sympathetic response resulting an increase in heart rate, blood pressure, and electrodermal activity as well as the activation of the hypothalamic–pituitary–adrenal (HPA) axis, leading to a slower release of cortisol (for an overview see Starcke & Brand, 2016). On the *psychological level*, stress is usually connected to affective responses, which can range from more arousal-related feelings such as tension or nervousness to feelings that involve fear, anxiety, frustration, sadness, and anger and even positive emotional states (Lazarus, 2006).

Although physiological and psychological stress responses such as cortisol or cardiovascular reactivity and negative affect often go hand in hand, they can also appear independently (e.g., Campbell & Ehlert, 2012; Dickerson & Kemeny, 2004). For instance, people may differentially interpret the arousal induced by a stressor depending on how they appraise the situation—with some perceiving a stressor as an exciting challenge while others perceive it as a threat (Lee & Andrade, 2015). Furthermore, the temporal trajectories of subjective and physiological responses differ. Negative subjective feelings and symptoms of sympathetic activation tend to be most intense in the presence of the stressor but decrease once the stressor has passed (McRae et al., 2006). In contrast, for instance, cortisol levels may reach their peak only after a (short-lived) stressor has passed and can take hours to fall back to baseline levels (McRae et al., 2006). Accordingly, depending on the nature of the stressor and its appraisal by the person, the timing, and previously existing moods or emotions, the pattern of physiological and psychological stress responses may differ.

1.2 | Stress responses and risky decision making

As reviewed above, the literature on the influence of acute stress on risky decision making is rather heterogeneous, with studies finding increased risk seeking, increased risk aversion, and no effect. A recent meta-analysis found that overall, acute stress leads to more risky decisions (Starcke & Brand, 2016). However, the authors found no relation between single measures of physiological stress responses (cortisol and α-amylase) and risky decision making.

Similar to the literature on stress, research on the relation of affective responses and risky decision making has resulted in inconsistent results (for a review see Cohen, Pham, & Andrade, 2007). Although the majority of research has suggested that negative emotions such as sadness or fear (but not anger) decrease risky decision making (e.g., Chou, Lee, & Ho, 2007; Kamstra, Kramer, & Levi, 2003; Stanton, Reeck, Huetel, & Labar, 2014; Yuen & Lee, 2003), several studies have reported the opposite pattern (e.g., Kliger & Levy, 2003; Mittal & Ross, 1998; Tice, Bratslavsky, & Baumeister, 2001).

Here, we suggest that some of the inconsistencies in the effect of stress (and also negative affect) on risky decision making may be caused by focusing on a single stress response (e.g., cortisol or affect) and neglecting the potential interplay between physiological and psychological responses to acute stressors. Two strands of research motivate this idea: First, Starcke and Brand (2016) found a difference between systemic stressors, physiological stressors such as pain, and processive stressors, stressors that involve an interpretation of the environment (Dedovic, Duchesne, Andrews, Engert, & Pruessner, 2009), in their meta-analysis. The most commonly used systemic stressor was the CPT, whereas the most frequently used processive stressor was the TSST. The CPT and TSST, however, also differ in the degree to which they elicit negative affective responses. In contrast to the TSST, the CPT frequently does not elicit a lasting negative affective response (McRae et al., 2006). The SECPT is rated as similarly unpleasant as the CPT (Nowacki et al., 2019; Schwabe et al., 2008) and leads to decreases in positive mood

and calmness immediately after the stress induction (e.g., Schwabe, Höffken, Tegenthoff, & Wolf, 2013), but does not seem to lead to a longer lasting mood disturbance (Giles, Mahoney, Brunye, Taylor, & Kanarek, 2014). Accordingly, differences in the impact of these stressors on risky decision making may also depend on the level of negative affect participants experienced. Second, research on the impact of negative affective states on risky decision making has suggested that increased risk taking is more likely when participants not only feel negative affect but also report high levels of arousal (Leith & Baumeister, 1996), because under these condition people (a) are motivated to seek rewards to repair their affective state and (b) have reduced capacity for self-regulation (e.g., Garg, Wansink, & Inman, 2007; Tice et al., 2001). Here, we investigated how psychological and physiological responses interact in their influence on risky decision making in an exploratory empirical study.

2 | METHOD

In the study we investigated financial risky decision making under stress and no stress while measuring physiological (cortisol and α-amylase levels, blood pressure) and subjective affective responses (self-reported valence and arousal). We induced stress with a CPT that contained a social evaluative element. Risk taking was measured with a behavioral decision task that consisted of 40 decisions between two gambles that contained positive and negative outcomes. The study was approved by the ethical committee of the cantons *Basel* and *Basel-land* (EKBB; Ref No. 181/11).

2.1 | Design

We implemented a mixed experimental design in which we examined the influence of stress on risk taking within and between participants. In a treatment group (*stress condition*) we observed risk taking before and after a stress induction. In addition, we compared risk taking of the treatment group with risk taking in a control group (*no stress condition*) in which the stress induction was replaced with a neutral manipulation. Cortisol, α-amylase, and subjective affect were measured at four time points: before the first risk-taking task (T1), after the first risk-taking task (T2), after the stress induction but before the second risk-taking task (T3), and after the second risk-taking task (T4). Blood pressure was measured at five time points: baseline, 30 s, 3 min, 8 min, and 20 min after the start of the stressor. Figure 1 presents a schematic overview of the experimental procedure. The data can be found on OSF. https://osf.io/dmzku.

2.2 | Participants

Seventy participants (40 in the stress condition and 30 in the no-stress condition, M_{age} = 24.4 years, SD_{age} = 5.3) were recruited at the University of Basel. Participants could only take part in the study if they did not take medication including corticoids (except oral contraceptives), did not habitually smoke, and had no psychological or physiological diagnoses. In about one fourth to one third of participants even reliable acute stressors such as the TSST do not result in an increase in cortisol (Kirschbaum et al., 1993). Thus, we collected more participants in the stress condition, to ensure that the stress manipulation would lead to an increase in cortisol for a sufficient sample of participants. Forty-eight participants identified as females and 22 as males. Fourteen males (35%) were in the stress condition and 8 (27%) in the control condition, $\chi^2(1) = 0.55$, $p = .604$. Mean age of participants in the control condition was 26 and 23 years in the stress condition, $t(42.63) = 1.83$, $p = .075$. Participants received 20 Swiss francs (CHF) per hour as compensation for participating in the study (approx. USD 22). Additionally, one of the decisions was randomly chosen and the preferred gamble was played. Participants received/paid 10% of the gamble's

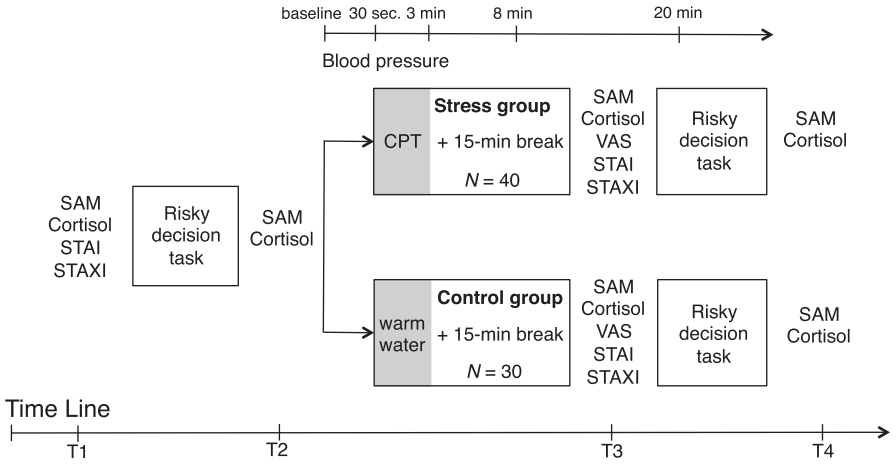

FIGURE 1 Overview of the experimental design and the experimental time line. T1–T4 indicate the measurement time points for cortisol and subjective affect (T2 was on average 17 min, T3 on average 40 min, and T4 on average 51 min after T1). *N* = sample size. CPT, cold pressor task; SAM, self-assessment manikin; STAI, state-trait anxiety inventory; STAXI, state-trait anger expression inventory; VAS, visual analogue scale

outcome. Overall, testing took 1 hr, 30 min. Testing took place in the afternoon between 12 noon and 3 p.m. Informed consent was obtained from all participants. Technical problems and an experimenter error resulted in a total of eight missing data points (one data point each on the following variables: baseline diastolic blood pressure (DBP), diastolic and systolic blood pressure (SBP) at 3 min and 8 min, α-amylase at measurement time points 3 and 4, time passed between measurement time point 3 and 4). We used multiple imputation to obtain 10 estimates for each of these values based on a predictive mean matching algorithm with 50 iterations as implemented in IBM SPSS Statistics version 23 using all physiological measurements as well as gender, condition, and the measurement time points as predictors. We then imputed the mean of the predicted values. In addition, we also conducted analyses excluding participants with missing values pairwise from the analyses. The findings only deviate slightly from the results with imputed missing values and all conclusions hold. A full report of these analyses can be found in the supplemental online materials on OSF.

An outlier analysis indicated that three participants (one in the control and two in the stress condition) had extremely high (>3 *SD*) cortisol values and one participant had (in the control condition) extremely high α-amylase values during the first or second measurements. Accordingly, we excluded these participants pairwise, that is just from the analyses, in which measures of cortisol or α-amylase were included.[1]

2.3 | Financial decision-making task

The financial decision-making task consisted of 40 decisions between two gambles. Each trial consisted of a choice between a reference gamble (Gamble A), in which participants could win CHF 15 or lose CHF 5 with a probability of .5 (expected value of CHF 5), and a target gamble (Gamble B). The reference gamble was the same in every trial, but there were 40 different target gambles structured in two sets: (a) high-outcome gambles (e.g., win/lose CHF 60 with a probability of .5) and (b) low-outcome gambles (e.g., win/lose CHF 30 with a probability of .5). For each gamble type (high or low outcome) we created sets of gambles by varying the target gamble's expected value from −5 to 15/30 in steps of CHF 5. The expected value was varied by changing either (a) the amount that could be won, (b) the amount that could be lost, or (c) the probability with which each outcome could occur (see Table 1 for an overview). The financial decision-making task essentially follows the logic of multiple-price lists that have been widely used in economics (e.g., Pedroni et al., 2017).

The order in which the target gambles were presented was randomly determined for each participant. Reference and target gambles were presented sequentially. Each trial started with a fixation cross (100 ms). Then the reference gamble was presented until participants pressed the return key. The target gamble appeared until participants made a choice by pressing "1" for the reference gamble or "2" for the target gamble. The task was implemented in Presentation (Neurobehavioral Systems, Inc., Berkeley, CA). A screenshot of the presentation of a target gamble can be found in the online Supporting Information on OSF. https://osf.io/dmzku

2.4 | Stress manipulation and measurements

In the stress condition, we used a variation of the CPT (Hines & Brown, 1936; Lovallo, 1975). The CPT is a frequently used method to induce a stress response. In the CPT participants immerse their right hand in ice water (0–4°C, $M = 1.86$°C, $SD = 0.67$) for as long as possible up to 3 min. It is a commonly used systemic stressor that usually does not influence participants' affective valence. Because it has been shown that physiological stress responses are increased when a social evaluative element is added to the CPT, we used an adaptation of the SECPT (Schwabe et al., 2008). In our task the experimenter watched the participants during the task, but we did not film participants or tell them that their facial expressions would be analyzed. We chose this version for simplicity and as we expected it to reliably induce stress while keeping effects on affective valence to a minimum.

Five participants had to remove their hand earlier because it was too painful. During the CPT the experimenter was situated in the same room as the participant, sitting slightly behind the participant, monitoring the CPT and blood pressure.[2] A female and a male experimenter, who

[1]Including the participants with outliers in the respective analyses leads to the same conclusions for all analyses with cortisol and only small changes in the estimated parameter values. For instance, in the analyses with cortisol reactivity as a moderator variable the interaction between affective valence and cortisol reactivity is also significant, $\Delta R^2 = .08$, $F(1,66) = 6.60$, $p = .013$ and we find an effect of affect when cortisol reactivity was high (+ *SD*), $b = -1.32$, $SE = 0.65$, $p = .045$. For α-amylase, including the outlier, we find a significant interaction between α-amylase and affective valence, $\Delta R^2 = .08$, $F(1,66) = 4.24$, $p = .044$, but the effect of affective valence on change in risk taking when α-amylase was high (+ *SD*) remains non-significant, $p = .088$.

[2]The stress induction in these five participants was successful as can be seen in a strong cortisol response (i.e., cortisol levels at T3 were $M = 29.92$, $SD = 8.68$) and thus we included them in the analyses. To investigate whether these participants unduly influenced the results we also conducted analyses excluding them. These analyses showed the same pattern of results as reported for the complete sample: The stress group had significantly higher values than the control group for cortisol and anxiety at T3 and SBP and DBP 3 min after the start of the CPT. Furthermore, also the moderation analysis of affective valence and stress showed the same pattern of results with a significant interaction ($p = .008$) and an effect of affective valence on risk taking in the stress condition, $b = -1.29$, $SE = 0.62$, $p = .041$.

TABLE 1 Overview of the target gambles

Gamble no.	*p* (Gain)	Gain	*p* (Loss)	Loss	EV	Set
1	.50	60	.50	−70	−5	High
2	.50	60	.50	−60	0	High
3	.50	60	.50	−50	5	High
4	.50	60	.50	−40	10	High
5	.50	60	.50	−30	15	High
6	.50	60	.50	−20	20	High
7	.50	60	.50	−10	25	High
8	.50	30	.50	−40	−5	Low
9	.50	30	.50	−30	0	Low
10	.50	30	.50	−20	5	Low
11	.50	30	.50	−10	10	Low
12	.50	30	.50	−0.1	15	Low
13	.50	50	.50	−60	−5	High
14	.50	60	.50	−60	0	High
15	.50	70	.50	−60	5	High
16	.50	80	.50	−60	10	High
17	.50	90	.50	−60	15	High
18	.50	100	.50	−60	20	High
19	.50	110	.50	−60	25	High
20	.50	120	.50	−60	30	High
21	.50	20	.50	−30	−5	Low
22	.50	30	.50	−30	0	Low
23	.50	40	.50	−30	5	Low
24	.50	50	.50	−30	10	Low
25	.50	60	.50	−30	15	Low
26	.50	70	.50	−30	20	Low
27	.50	80	.50	−30	25	Low
28	.50	90	.50	−30	30	Low
29	.46	60	.54	−60	−5	High
30	.54	60	.46	−60	5	High
31	.58	60	.42	−60	10	High
32	.63	60	.37	−60	15	High
33	.67	60	.33	−60	20	High
34	.71	60	.29	−60	25	High
35	.75	60	.25	−60	30	High
36	.42	30	.58	−30	−5	Low
37	.58	30	.42	−30	5	Low
38	.67	30	.33	−30	10	Low
39	.75	30	.25	−30	15	Low
40	.83	30	.17	−30	20	Low

Note: *p* (Gain) = probability of receiving the positive outcome (Gain); *p* (Loss) = probability of receiving the negative outcome (Loss).

Abbreviation: EV, gamble's expected value.

were randomly assigned to participants, conducted the CPT. Participants' stress responses did not differ between the two experimenters (for similar findings, see Schwabe & Schächinger, 2018). The control condition followed the same procedure with the only differences that participants immersed their hand in warm water (37–40°C, $M = 38.98$°C,

SD = 0.81). During the water task we measured systolic and diastolic blood pressure at five measurement points: baseline, 30 s, 3 min, 8 min, and 20 min after the start of the task to record the cardiovascular response and to abort the experiment in case of dangerously high or low levels of blood pressure. Blood pressure was measured with a sphygmomanometer on the left upper arm.

As subjective stress responses we measured affective valence, arousal, and dominance (measuring how self-confident and in control participants feel) with the Self-Assessment Manikin (SAM; Hodes, Cook, & Lang, 1985) using a 9-point scale at T1 to T4 (see Figure 1). The SAM consists of five drawings, each depicting different states of affective valence (1 = very happy to 9 = very sad), arousal (1 = high arousal to 9 = relaxed and calm), and dominance/self-confidence (1 = unsure/not in control to 9 = dominant and in control). Participants were asked to select the picture that described their current affective state best or to indicate between which pictures their current affect lay. Additionally, we measured current anxiety and anger with the State-Trait Anxiety Inventory (STAI; Laux, Glanzmann, Schaffner, & Spielberger, 1981) and the State-Trait Anger Expression Inventory (STAXI; Schwenkmezger, Hodapp, & Spielberger, 1992) at T1 and T3.

Last, we collected saliva samples with Salivettes (Sarstedt, Nuembrecht, Germany) to determine cortisol and α-amylase levels at T1 to T4. Saliva samples were analyzed at the laboratory of the Technical University Dresden, Germany. Salivary free cortisol levels were determined using a chemiluminescence immunoassay (IBL International GmbH, Hamburg, Germany) with intra- and interassay precision of 2.5% and 4.7%, respectively.

2.5 | Procedure

After participants arrived we determined if they met the inclusion criteria for the study, obtained informed consent, and gave them approximately 8 oz of water to drink. Then, we took the first saliva sample and measured affective valence and arousal (T1, see Figure 1). After the measurements participants immediately proceeded with the first session of the financial decision-making task. Thereafter, participants gave the second saliva sample and again completed the affective valence and arousal measures (T2). Next, participants proceeded with the stress manipulation. After the stress manipulation participants indicated on a visual analog scale ranging from not unpleasant at all (0) to very unpleasant (20) how unpleasant the CPT had been. We took the third saliva sample 15 min after the stress manipulation and measured affective and arousal (T3). Immediately afterward participants performed the financial risk-taking task again (Session 2). After that we again measured affective valence and arousal and took the fourth saliva sample (T4). In addition, participants filled out a questionnaire measuring novelty seeking as a potential correlate of risk attitudes (Cloninger, Przybeck, Svrakic, & Wetzel, 1994).[3]

3 | RESULTS

3.1 | Valence, arousal, and physiological stress responses

Analyses were conducted with IBM SPSS Statistics version 23. First we analyzed if the stress manipulation influenced participants' subjective responses and physiological responses (for means and *SDs* see Tables 2 and 3) using analyses of variance (ANOVAs) on valence, subjective arousal, dominance, SBP, DBP, α-amylase, and cortisol with measurement time (T1–T4 for cortisol and subjective arousal and the five measurement points for systolic and DBP) as within-subject factors and stress condition as a between-subjects factor. When necessary we adjusted the degrees of freedom according to the Greenhouse–Geisser correction. The analyses showed that subjective arousal, SBP, DBP, and cortisol levels increased in the stress group but not in the control group. In the control group, cortisol, SBP, and arousal decreased, suggesting that participants' initial tension decreased during participation. This was indicated by significant interactions of measurement time and stress condition, arousal: $F(2.49, 169.44) = 4.911$, $p = .003$, partial $\eta^2 = 0.07$; SBP: $F(3.04, 206.78) = 18.71$, $p < .001$, partial $\eta^2 = 0.22$; DBP: $F(3.52, 239.34) = 34.36$, $p < .001$, partial $\eta^2 = 0.34$; cortisol: $F(1.39, 90.1) = 26.31$, $p < .001$, partial $\eta^2 = 0.29$; see Figure 2a. Additional *t* tests revealed that participants in the stress and control group did not differ in cortisol levels at the first two measurement points (all *p*s > .84) but that the stress group had higher cortisol levels at T3 and T4 (all *p*s < .001). Similarly, subjective arousal did not differ significantly between the two groups at the start of the study (T1, *p*s > .12), but the stress group showed higher arousal after the stress manipulation at T3, $t(68) = 2$, $p = .05$ (see Table 2 for means and *SD*s). For SBP, participants had somewhat higher values in the control group than the stress group at baseline, $t(68) = 2.25$, $p = .028$, but did not differ 30 s after immersing their hand in the water, $t(68) = 0.88$, $p = .381$. In regard to the measurements 3, 8, and 20 min after putting their hand into the water, participants in the stress group had clearly higher values than participants in the control group after 3 min, $t(68) = -4.22$, $p < .001$, somewhat higher values after 8 min, $t(66.35) = -1.71$, $p = .09$, and no difference

[3]Participants' eye movements were also recorded in this study. However, we do not report analyses on eye tracking or novelty seeking here, as they are not the focus of this manuscript.

TABLE 2 Descriptive statistics (means and *SD*s) for measures of physiological and affective stress responses and risk taking by stress condition

Measure	T1	T2	T3	T4
Stress condition				
Risk premium	10.53 (5.59)		9.88 (5.85)	
Physiological measures				
Cortisol (nmol/l)	10.69 (6.00)	9.02 (5.05)	17.57 (10.84)	19.08 (12.38)
α-amylase (units/ml)	29.56 (21.45)	38.14 (31.25)	35.24 (28.59)	40.77 (36.29)
Psychological measures				
Valence	2.85 (1.00)	3.08 (1.19)	2.95 (1.11)	3.03 (1.10)
Arousal	6.38 (1.29)	6.38 (1.63)	6.03 (1.95)	6.90 (1.28)
Dominance	5.95 (1.20)	5.85 (1.10)	6.13 (1.18)	6.23 (1.14)
State anger	1.12 (0.25)	–	1.09 (0.19)	–
State anxiety	1.83 (0.28)	–	1.78 (0.31)	–
Control condition				
Risk premium	9.99 (6.91)		9.80 (7.86)	
Physiological measures				
Cortisol (nmol/l)	10.96 (4.64)	9.10 (3.76)	7.77 (3.29)	7.05 (2.87)
α-amylase (units/ml)	27.72 (19.98)	41.69 (26.49)	28.95 (18.80)	33.39 (24.39)
Psychological measures				
Valence	3.17 (1.09)	3.03 (1.16)	3.07 (1.31)	2.93 (1.17)
Arousal	6.00 (1.55)	6.17 (1.70)	6.90 (1.60)	6.83 (1.88)
Dominance	6.10 (1.35)	6.27 (1.23)	6.43 (1.33)	6.53 (1.43)
State anger	1.10 (0.15)	–	1.07 (0.12)	–
State anxiety	1.72 (0.30)	–	1.58 (0.27)	–

Note: $N_{\text{stress group}} = 40$; $N_{\text{control group}} = 30$ except for cortisol where $N_{\text{stress group}} = 38$ and $N_{\text{control group}} = 29$ and for α-amylase where $N_{\text{control group}} = 29$; higher numbers indicate more negative valence, less arousal, more dominance/control, more anger, and more anxiety.

TABLE 3 Descriptive statistics for systolic and diastolic blood pressure (means and *SD*s) in the two conditions

Condition	Baseline	30 s	3 min	8 min	20 min
Stress Condition					
SBP	120.43 (12.12)	120.58 (11.99)	134.53 (13.72)	127.39 (19.67)	118 (10.41)
DBP	69.55 (6.37)	70.93 (7.00)	88.57 (10.65)	74.91 (10.61)	70.48 (7.34)
Control Condition					
SBP	126.83 (11.88)	123.27 (13.45)	121.23 (12.12)	120.80 (12.46)	118.13 (12.10)
DBP	76.80 (7.20)	75.53 (8.68)	75.97 (8.56)	73.57 (6.68)	73.80 (5.57)

Note: $N_{\text{stress}} = 40$; $N_{\text{control}} = 30$.

Abbreviations: DBP, diastolic blood pressure; SBP, Systolic blood pressure.

after 20 min, $t(68) = 0.05$, $p = .961$; see Table 3 for means and *SD*s. Average DBP was higher in the control condition than in the stress condition at baseline and after the first 30 s of the CPT (Baseline: $t(68) = 4.46$, $p < .001$; 30 s.: $t(68) = 2.46$, $p = .017$, see Table 3). In the stress condition DBP increased and was significantly higher than in the control condition after 3 min, $t(68) = -5.32$, $p < .001$. Afterwards DBP decreased again in the stress group and was similar to the control group after 8 min, $t(68) = -0.61$, $p = .55$, and lower after 20 min, $t(68) = 2.07$, $p = .042$. The analysis of α-amylase activity did not reveal a significant interaction between measurement time and condition, $F(2.20,147.13) = 2.28$, $p = .101$, partial $\eta^2 = 0.03$. Participants reported the water immersion task as much more unpleasant in the stress condition ($M = 15.43$, $SD = 4.55$) than in the control condition ($M = 3.10$, $SD = 4.20$), $t(68) = 11.58$, $p < .001$.

In sum, the stress manipulation successfully induced subjective and physiological stress. In contrast, an analysis of

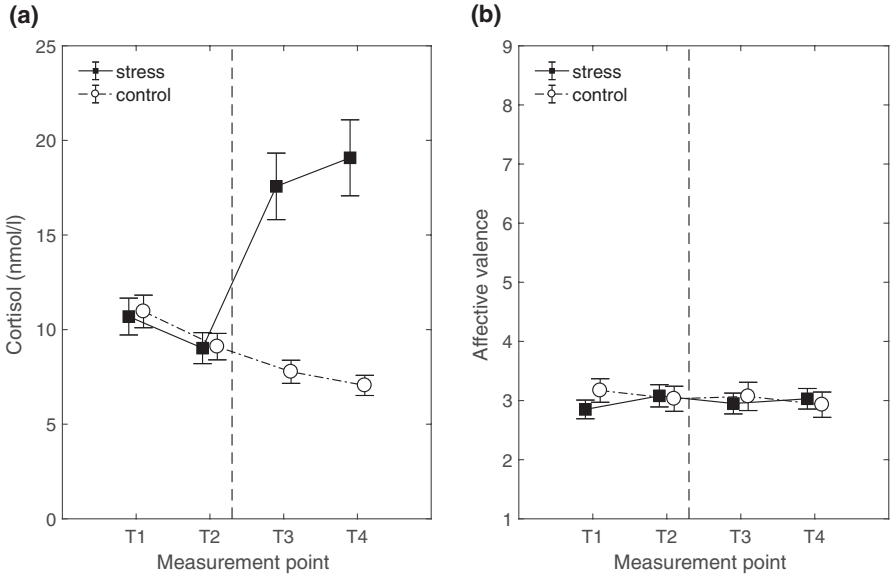

FIGURE 2 (a) Average cortisol levels (nmol/l) and (b) affective valence over the time course of the study for the control group and the stress group. T1–T4 denote the four measurement time points. The vertical line indicates the stress manipulation taking place between T2 and T3. Error bars denote one standard error

affective valence showed that the stress manipulation did not have an effect on affective valence. As illustrated in Figure 2b, there was no interaction between measurement time and stress condition, $F(3, 204) = 1.02$, $p = .385$, partial $\eta^2 = 0.02$, nor main effects of time or stress condition (all $ps > .65$). Additional t tests revealed that the stress and the control groups did not differ in their affective valence at any of the measurement points (all $ps < .21$). There were also no main effect of stress condition and no interaction of stress condition and measurement time point for state anger or dominance (all $ps > .27$).

To assess the pattern of physiological stress responses and affect, we correlated measures of α-amylase activity, cortisol, and SBP and DBP reactivity with measures of subjective arousal and valence and their change from T2 to T3. Cortisol and α-amylase reactivity were measured as the area under the curve with respect to increase starting from T2 using the trapezoid formula (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003), and SBP and DPB reactivity as the increase in SBP/DPB during the water immersion task (increase between 30 s and 3 min after the start). SBP reactivity was significantly correlated with DBP reactivity, $r(70) = .75$, $p < .001$, change in reported arousal, $r(70) = -.428$, $p = .020$, cortisol reactivity, $r(67) = .55$, $p < .001$, and α-amylase activity, $r(69) = .32$, $p = .010$, but not with change in affective valence or affective valence at T3. Cortisol reactivity correlated with DBP reactivity, $r(67) = .53$, $p \leq .001$, and α-amylase activity, $r(66) = .32$, $p = .009$, but did not correlate significantly with change in arousal, $r(67) = -.19$, $p = .117$, affective valence at T3, $r(67) = .01$, $p = .924$, or change in affective valence, $r(67) = .04$, $p = .780$. Similarly, DBP reactivity and α-amylase activity were correlated, $r(69) = .39$, $p = .001$, but did not correlate significantly with the subjective measures. Arousal at T3 was related to valence at T3, $r(70) = -.43$, $p < .001$.

3.2 | Risky decision making

We used multiple-price lists to access people's risk preferences (e.g., Pedroni et al., 2017). The gambles were constructed so that the target gambles were more risky (i.e., had a higher variance) than the reference gamble, in which people could gain 15 or lose 5 with a probability of .5. Within each of the six sets the EV of the first target gamble was lower than the EV of the reference gamble (i.e., −5 compared to the EV of 5 for the reference gamble) and then increased in steps of 5, making the target gamble more and more attractive in comparison to the reference gamble (see Table 1). We assessed a person's risk preference by calculating a risk premium that measured how much higher the EV of a target gamble needed to be than the EV of the reference gamble for that person to prefer the target gamble to the reference gamble. Specifically, we determined in each set the target gamble with the lowest EV that was preferred to the reference gamble, subtracted the EV of the reference gamble from its EV, and calculated the mean across the sets. For instance, a person who still preferred the reference gamble to the target gamble "gain 50, lose 30 with $p = .5$" with an EV of 10, but accepted the target gamble "gain 60, lose 30 with $p = .5$" with an EV of 15 would have a risk premium of 5. Accordingly, positive risk premiums reveal risk aversion and negative scores indicate risk seekingness.[4] On average

[4]If a person never chose the target gamble in a set the risk premium was set as the highest EV in the respective set. If a person always chose the target gamble in a set the risk premium score was set at −10, that is, lower than the lowest EV in the set. If people switched more than once in a set we determined the target gamble with the lowest EV that was preferred to the reference gamble and subtracted the EV of the reference value from its EV. Then we determined the target gamble with the highest EV that was rejected in favor of the reference gamble and took its EV. The risk premium was calculated as the mean of these two values. The conclusions do not change if other measures of risk taking are used, such as the proportion of target gamble choices.

participants had a positive risk premium of $M = 10.30$ ($SD = 6.15$) in the first session and $M = 9.85$ ($SD = 6.73$) in the second session, indicating that participants were risk averse. To assess the reliability and internal consistency of the risk premium we correlated risk premiums measured for the high and low variance gambles. This revealed a good consistency of $r = .79$. A further analysis considering the frequency with which people were switching more than once in a set showed that in both, the control and the stress condition, people responded more consistently in the second session, that is, less frequently switched several times within a set.

To analyze whether the stress manipulation influenced participants' risk preferences we ran an ANOVA on the average risk premium with session (before/after the stress induction) as the within-subject factor and stress condition as the between-subjects factor. We did not find a main effect of session, $F(1,68) = 0.97$, $p = .328$, or stress, $F(1,68) = 0.04$, $p = .837$, nor an interaction between them, $F(1,68) = 0.29$, $p = .59$ (for means and *SD*s see Table 2), suggesting no direct effect of the stress manipulation on financial risk taking. These results did not change when controlling for base level systolic and DBP.

Because previous studies have shown that men and women react differently to stress (e.g., Lighthall, Mather, & Gorlick, 2009), we ran additional analyses to investigate if gender influenced the effect of stress on risk taking. Although men had slightly lower risk premiums, we did not find a significant effect of gender on risk premiums ($M_{men} = 8.11$, $SE = 1.37$, $M_{women} = 10.93$, $SE = 0.90$), $F(1,66) = 2.99$, $p = .089$, partial $\eta^2 = 0.04$. Also, gender did not interact with stress or measurement time point (all *p*s > .42) and did not significantly influence cortisol reactivity, α-amylase reactivity, SBP and DBP reactivity or measures of arousal and affect and thus was not considered in the subsequent analyses.

3.3 | Relating physiological and affective measures to risky decision making

Next, we investigated whether physiological stress responses and affective measures were related to risky decision making. To examine the relation between affective measures and risky decision making we correlated measures of affect and arousal before the risky decision-making task with the risk premiums. Affective valence at T1 was negatively correlated with risk premiums in Session 1, $r(70) = -.25$, $p = .034$, but affective valence at T3 did not correlate with risk premiums in Session 2, $r(70) = -.13$, $p = .290$, suggesting more negative affect was related to somewhat less risk averse decisions in the first session, but not after the stress manipulation. Arousal did not correlate with risk premiums at either time point, *p*s > .38.

For cortisol, neither at T1 nor at T3 were cortisol levels correlated with risk premiums before and after the stress manipulation (all *p*s > .50); nor was cortisol reactivity related to risk premiums after the stress induction, $r(67) = .09$, $p = .466$, or change in risk premiums, $r(67) = .12$, $p = .337$. Risk premiums and change in risk premiums was also not significantly related to SBP, DBP or α-amylase reactivity. These results did not change when controlling for base levels of cortisol, α-amylase, and SBP and DBP, respectively.

3.4 | The joint influence of affective valence and physiological stress responses on risky decision making

Finally, we explored whether physiological and psychological responses, specifically affective valence, interacted in their influence on risky decision making. In particular, we considered whether risk preferences depended on the joint influence of affective valence and physiological measures. For this analysis we focused on affective valence at T3, that is how positive or negative participants felt before completing the second risky decision-making task. We focused on affective valence because previous research has indicated that it is related to risky decision making and may interact with levels of arousal (Leith & Baumeister, 1996). We chose affective valence at T3 because it best reflects participants' positive and negative affect when making the decision under stress or no stress.[5] An ANOVA predicting risk premiums with measurement time point (before and after the stress manipulation) as the within-subject factor and stress condition, affect valence after the stress induction, and their interaction as between-subject factors showed a significant three-way interaction between measurement time point, stress condition, and affect, $F(1, 66) = 9.36$, $p = .003$, partial $\eta^2 = 0.12$. To confirm this result and to investigate it more closely, we conducted a moderated regression with change in risk premiums from Session 1 to Session 2 as the dependent variable, affect after the stress induction as the independent variable, and stress condition as the moderator variable using the SPSS macro by Hayes (2013), a tool to facilitate calculating a moderated regression in SPSS, centering affect and stress and using SE-consistent estimators. As illustrated in Figure 3, the analysis confirmed the interaction between stress condition and affect, $b = -2.08$, $SE = 0.74$, in the direction that in the

[5]An alternative choice would be to use a measure of affective reactivity (i.e., a change in affect from T1 to T3 similar to the measures of physiological stress reactivity). We focus here on affective valence at T3, because most theories on affect and risky decision making consider the absolute level of affect to influence decision making and rarely consider change in affect.

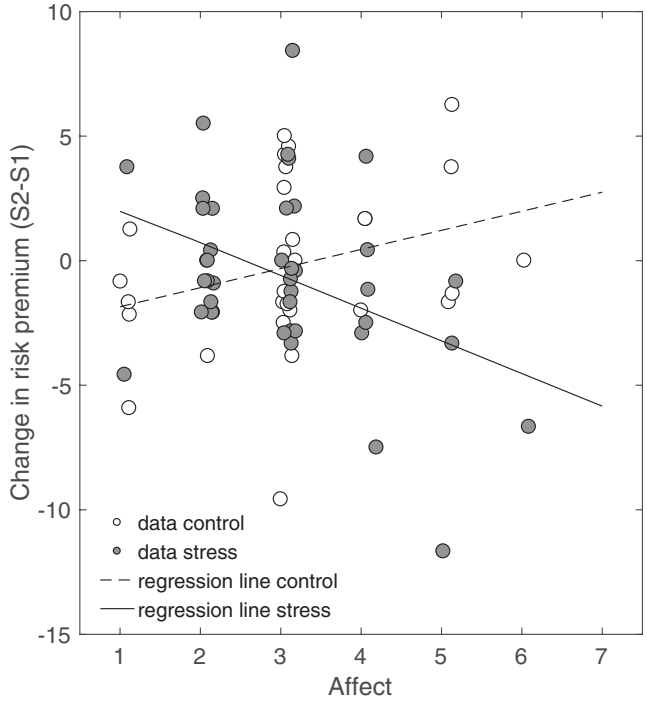

FIGURE 3 Scatterplot with regression lines depicting the estimated relation between the change in risk premiums between the first (S1) and the second (S2) session and affect measured before S2 in the control condition (white circles, dotted line) and the stress condition (grey circles, black line). Higher scores in change in risk premiums indicate an increase in risk aversion from S1 to S2. Higher scores on affect indicate more negative affect

stress condition—but not in the control condition—negative affect predicted a decrease in risk premiums: That is, participants became more risk seeking in Session 2. Including the interaction increased the explained variance significantly compared to a model without interaction, $\Delta R^2 = .12$, $F(1,66) = 7.93$, $p = .006$. Separate tests of the influence of affect in the stress and the control condition indicated that in the stress condition more negative affect led to a significant decrease in risk premiums, $b = -1.31$, $SE = 0.60$, $p = .034$. In contrast, in the control condition the relation between negative affect and change in risk premiums was non-significant but pointed in the opposite direction: That is, more negative affect led to an increase in risk premiums, $b = 0.77$, $SE = 0.42$, $p = .075$. Further analyses controlling for baseline affect and risk premiums in Session 1 led to the same conclusions. Similarly, controlling for diastolic and SBP at baseline, where we had found differences between the control and the stress group, did not change the pattern of results (interaction: $p = .005$; effect of affect valence in stress group: $b = -1.41$, $SE = 0.69$, $p = .044$).

To analyze whether the interaction between stress and affective valence was related to physiological stress responses, we ran additional analyses, in which we used physiological stress reactivity scores of all participants (i.e., cortisol, SPB, DPB, and α-amylase reactivity), as the respective moderator variable instead of stress condition. Only the analysis with cortisol reactivity indicated a similar moderation: Negative affect was related to a decrease in risk premiums in participants with high cortisol reactivity, $\Delta R^2 = .07$, $F(1,63) = 5.17$, $p = .026$; 1 *SD* over the mean cortisol reactivity, $b = -1.37$, $SE = 0.68$, $p = .048$, but not in participants with average cortisol reactivity or a decrease in cortisol levels. The analyses for DPB and α-amylase activity indicated a similar pattern, but the effects were not statistically significant.

4 | DISCUSSION

Research on the effects of stress on risk taking has produced a mixed set of results. In the current research we investigated whether considering the interplay of physiological and psychological stress responses could contribute to understanding when stress will increase risky financial decision making. To this end, we measured participants' physiological and psychological reactions to an acute stressor as well as their risk preferences.

The stress manipulation led to increased cardiovascular and cortisol reactivity as well as self-reported arousal but did not influence self-reported affective valence. The different physiological stress measures were moderately correlated but unrelated to affective valence. These results suggest that the manipulation of acute stress was successful, but show that not all stress induction methods exert a longer lasting effect on affective valence. They resonate with research, suggesting that the TSST induces longer lasting negative affect, whereas the CPT and SECPT do not (Giles et al., 2014; McRae et al., 2006).

On average, the stress manipulation did not influence risk preferences. Also, none of the physiological stress responses, that is, cortisol, α-amylase, and SBP reactivity, showed a direct and independent influence on risky decisions, as suggested by the non-significant zero-order correlations between all measures and risk premiums. However, our results suggest that to understand how stress influences decision making it is necessary to consider the interplay of affect and physiological stress responses: After the stress manipulation, participants who reported high levels of negative affect became more risk seeking in the stress condition, whereas in the control group negative affect was not related to risky decision making—if anything, people in a negative affective state became less risk seeking in the second session. The same interaction effect was found when comparing participants with high and low cortisol reactivity. In participants who showed high increases in cortisol, negative affect was related to an increase in risky decision making in the second decision-making session,

but not in participants with no increase in cortisol. In the following we first review how our results relate to previous studies on stress and risky decision making discussing the role of stressor type and gender and then outline why affect and stress responses may interact. Finally, we consider the limitations of our study.

4.1 | Effects of the stress manipulation on risky decision making

The meta-analysis by Starcke and Brand (2016) reviewing the literature up to that point found a reliable effect of stress inductions on risky decision making with *processive* stressors such as the TSST, but no effect of *systemic* stressors such as the CPT. Similarly, another recent study by Sokol-Hessner et al. (2016) did also not find an effect of CPT (without social evaluative elements) on risk taking. However, we did not find an effect of our stress manipulation on participants' risky decision making, even though we used a stress manipulation that contained social evaluative (i.e., processive) elements. There are several potential explanations for this. For one, we used only an adaptation of the SECPT with a reduced social evaluative element compared to the original version (Schwabe et al., 2008). Physiological stress responses are less pronounced in the CPT compared to the SECPT and the TSST (e.g., McRae et al., 2006; Nowacki et al., 2019; Schwabe et al., 2008). Thus, even though we found a reliable effect on cortisol and blood pressure, it is possibly the stress responses were not strong enough to elicit effects on risky decision making. Against this explanation speaks, however, that the meta-analysis by Starcke and Brand (2016) did not show a relation between cortisol levels and risk taking, suggesting that the strength of the stress response does not suffice to explain whether an effect on risk taking is found or not. A second possibility is that we induced stress, but the social evaluative element in our stress induction was too weak to activate the respective neural pathways, given that we deviated from the original version by Schwabe et al. (2008). Dedovic et al. (2009) proposed that physical stressors involve different biological mechanisms than psychological stressors. Specifically, they argued that psychological stressors influence activation in the orbitofrontal and medial prefrontal cortex (PFC) that are important for integrating perceptual information as well as monitor and control emotional states. The orbitofrontal PFC is also involved in risky decision making (Mohr, Biele, Krugel, Li, & Heekeren, 2010; O'Neill & Schultz, 2013) providing a potential pathway how psychological stressors impact risk taking (see also Porcelli & Delgado, 2017; Porcelli, Lewis, & Delgado, 2012). Given that the orbitofrontal PFC also plays an important role in affect, this links to the third possibility, that we did not find an overall effect of the stress manipulation, because the stress manipulation did not induce negative affect. In our task risk taking increased under stress, but only for participants who also reported being in a negative affective state. This could be related to the relatively weak social evaluative component of our stress manipulation. However, given that also the SECPT does not seem to induce a longer lasting effect on mood (Giles et al., 2014) it is also possible that it is not necessary that negative affect is induced by the stressor. Instead, it could be that whether more risk taking occurs under stress depends on the level of negative affect experienced by a person. Accordingly, also systemic stressors could increase risk taking when participants are in a negative mood. We will discuss why negative affect may interact with stress to induce risk taking below. To disentangle these possibilities, further research using processive and systemic stressors as well as measuring affect at the time of the risk-taking task, which is often not the case in studies focusing on physiological stress responses, will be necessary.

A further potential explanation for our null results is that the effect of stress may depend on gender. In our study, we did not find significant gender differences. However, this could have been caused by a lack of power due to the relatively small number of men in our study. A growing number of studies have reported that the impact of stress may differ for men and women (Cahlíková & Cingl, 2017; Kluen, Agorastos, Wiedemann, & Schwabe, 2017; Lighthall et al., 2009; Nowacki et al., 2019; van den Bos, Harteveld, & Stoop, 2009). These studies suggest that the effect of stress on risk taking in men and women may depend on the stressor. In this vein, Nowacki et al. (2019) found an interaction between the type of the stressor (SECPT and CPT) and gender. Whereas women made more risky decisions after the SECPT, risk taking increased for men after the CPT. Similarly, Lighthall et al. (2009) found increased risk taking in men after the CPT, Kluen et al. (2017) found that giving hydrocortisone increased risk taking in men but not in women and Cahlíková and Cingl (2017) reported that men became more risk averse after the TSST. Yet, it should be noted that the meta-analysis by Starcke and Brand (2016) did not find an effect of gender on risk taking for processive or systemic stressors and also Sokol-Hessner et al. (2016) did not find an effect on risk taking in men with the CPT. This suggests that future research is necessary to investigate the origin of gender effects of stress on risk taking.

4.2 | Affect, stress, and risky decision making

In the moderation analyses we found an interaction between the stress condition and whether negative affect was related to more or less risk taking. Why would stress increase risk taking when participants also were experiencing negative

affect, but negative affect without stress reduce risk taking? The idea that negative affect sometimes increases and sometimes decreases risky decision making corresponds to the heterogeneity reported in the literature. Many studies reported that negative affective states, in particular fear but also sadness, reduce risk taking—arguably because negative affective states lead to a more negative view of the world, which could lead to an increased risk perception (Kamstra et al., 2003; Lerner & Keltner, 2001; Stanton et al., 2014; Yuen & Lee, 2003). Yet other studies found the opposite effect, that is, negative affective states increased risk taking (Kliger & Levy, 2003; Mittal & Ross, 1998; Tice et al., 2001) arguably because people are using risky decisions to improve or repair their mood (Isen & Labroo, 2003). To integrate these two contradicting approaches, it has been proposed that whether negative affect increases or decreases risk taking depends on the degree to which the decision maker perceives that risk taking can be used to regulate affect (Cohen et al., 2007). For instance, Lee and Andrade (2015) found that the same fear manipulation was related to risk averse behavior in a stock investment task, but let to more risk seeking when the same task was framed as an exciting casino game. Acute stress may influence whether risk taking is perceived as a suitable regulatory strategy: For one, acute stress and stress-related increases in cortisol have been argued to increase the salience of rewards (Starcke & Brand, 2016). Increased reward salience, in turn, should make a high-risk option (that includes large rewards) more promising as a means for regulating affect. Second, stress and increases in cortisol arguably impair self-control (Maier, Makwana, & Hare, 2015). For instance, research showed that stress increases immediate gratification in monetary choices (Fields, Lange, Ramos, Thamotharan, & Rassu, 2014; Lu et al., 2014) and when selecting unhealthy but tasty foods (Epel, Lapidus, McEwen, & Brownell, 2001; Newman, O'Connor, & Conner, 2007).

Accordingly, people in a negative affective state without stress may focus on the negative consequences related to choosing high-risk options, inhibiting risk taking. In contrast, people in a negative affective state and under stress may see high-risk options as an opportunity to regulate affect, because physiological stress responses such as the release of cortisol increase the salience of the rewards and impair self-control processes that would otherwise inhibit choosing options with high potential losses. The results also resonate with a study finding an increase in risk taking relatively shortly after a stressor but not after a longer period of time (Bendahan et al., 2017). An explanation is that negative affective reactions are most pronounced during or right after a stressor has been experienced but then return to baseline, whereas physiological responses such as an increase in cortisol last longer (but see Pabst, Brand, & Wolf, 2013b for a different pattern of results).

On a biological level, it has been argued that affective states can modulate decision making via multiple neural pathways involving areas that are important for reward sensitivity such as the orbitofrontal cortex and the insula (Damasio et al., 2000; Lane, Reiman, Ahern, Schwartz, & Davidson, 1997, for a review see Phelps, Lempert, & Sokol-Hessner, 2014). Both these areas have also been connected to assessment of rewards and risk and are also affected by acute stress (Mather & Lighthall, 2012; Porcelli & Delgado, 2017; Porcelli et al., 2012). Thus, the interplay of affect and stress could influence emotion regulation and the perception of rewards and ultimately risk taking.

4.3 | Limitations

An important limitation in our study is that we only measured affective valence and not specific emotions. Thus, it is unclear which negative emotional states were predominant in our sample. Indeed, the reported negative affect is likely to be a mixture of feelings ranging from fearful to annoyed, displeased to frustrated and sad depending on participants preexisting affective states as well as their task experience. But, different negative emotional states such as anger, sadness, and fear can differ in their influence on decision-making processes (Frey, Hertwig, & Rieskamp, 2014; Lerner & Keltner, 2001; Lerner, Li, Valdesolo, & Kassam, 2015; Raghunathan & Pham, 1999). Frequently, it is argued that anger increases risk taking, while fear decreases it (Lerner & Keltner, 2001). The effects of sadness are more inconsistent. Raghunathan and Pham (1999) argued that sadness should increase risk taking but other studies have found that depressive symptoms (Kamstra et al., 2003) and sad events (Lepori, 2015) decreased risk taking. Thus, the effect of sadness may depend on the interpretation of the situation and the potential for mood regulation (Cohen et al., 2007) and the interpretation of the task (see also Lee & Andrade, 2015 for differential effects of fear). Thus, further research teasing apart how specific emotional states interact with physiological measures of stress is necessary.

Second, cortisol responses to stress have been shown to depend on the female ovulatory cycle and the use of contraceptives (e.g., Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999), which we did not control for in this study. We also did not measure participants body mass index (BMI). This introduces variability in the effect of the stress induction on cortisol levels, which could potentially have contributed to the null effect of cortisol reactivity on risk preferences. Third, our study is an exploratory analysis and focuses on a single type of stress induction, raising the question of whether the results can be generalized to other stressors. In particular, as we used an adapted version of the SECPT, it would be good to replicate our results following the protocol of the SECPT more closely as well as use other stressors such

as the TSST or the CPT. Finally, recent research reported that a cognitive load manipulation leads to more unsystematic risk taking (Olschewski, Rieskamp, & Scheibehenne, 2018). In a similar vein, stress could lead to more errors in decision making. Although we did not find evidence for increased inconsistent choices in the risky decision task we used, it is possible that acute stress could also lead to more mistakes in decision making, in particular when stressors reduce cognitive resources.

5 | CONCLUSIONS

In sum, our study provides a first step toward understanding the complex relation between physiological and psychological components in the influence of stress on risky decision making. It offers evidence that stress-related physiological changes such as increases in cortisol and affective states interact in shaping risky decision making. Only when people were stressed and in a negative affective state did stress lead to increases in risk taking. These results show that when aiming to understand the influence of stress on risky decision making, it is necessary to consider not only single measures of stress responses but also the complex interplay of different response types.

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CONFLICT OF INTEREST

None of the authors has a conflict of interest.

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